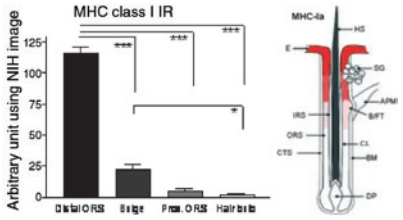
Evidence that the bulge region is a site of relative immune privilege in human hair follicles



Immune privilege (IP) describes a group of mechanisms that suppress local immune responses in a defined tissue site. Using immunohistochemistry, quantitative analysis and skin organ culture Meyer *et al.* show that the human hair follicle bulge also displays features of IP, i.e. downregulation of major histocompatibility complex (MHC) I, MHC II and β_2-microglobulin immunoreactivity, and upregulation of immunosuppressants (e.g. transforming growth factor-β2, α-melanocyte stimulating hormone, indoleamine-2,3-dioxygenase, macrophage migration inhibitory factor), CD200 and HLA-E. The role of bulge IP appears to be to protect the epithelial hair follicle stem cells in this area from immune attack. Therefore, loss of bulge IP may play a role in pathogenesis of primary cicatricial alopecia. *Br J Dermatol* 2008; 159:1077–85.

Hyperglycaemic conditions decrease cultured keratinocyte mobility: implications for impaired wound healing in patients with diabetes

Lan *et al.* have studied the effects of high glucose on keratinocyte proliferation and migration, as alterations in these processes may explain why poor wound healing is common in patients with diabetes. Keratinocytes were cultivated in normal and high glucose conditions. Their viability and mobility were evaluated. The mRNA expressions and activities of matrix metalloproteinase (MMP)-2 and MMP-9 were determined. The mRNA of their respective physiological inhibitors, tissue inhibitor of MMP (TIMP)-1 and TIMP-2, was also evaluated. Immunofluorescent staining and Western blotting were used to examine the expression of phosphorylated focal adhesion kinase ($pp125^{FAK}$). The impacts of high glucose on keratinocyte proliferation were assessed by 5-bromo-2′-deoxyuridine incorporation assay. High glucose treatment did not affect keratinocyte viability up to 3 days. In contrast, the mobility of keratinocytes, the activities and gene expressions of MMP-2 and MMP-9, the expression of $pp125^{FAK}$, and the cell proliferation after 5 days were significantly downregulated after hyperglycaemic treatments while the mRNA expression of TIMP-1 increased. Keratinocytes demonstrated reduced migration and decreased proliferation capacities under hyperglycaemic conditions. *Br J Dermatol* 2008; 159:1103–15.

The influence of antimalarial treatment on IL-1β, IL-6 and TNF-α mRNA expression on UVB-irradiated skin in systemic lupus erythematosus

Little is known about the cutaneous mechanisms of the antilupus effects of antimalarials. The influence was studied of 3 months of monotherapy with chloroquine on the skin mRNA expression of interleukin (IL)-1β, IL-6 and tumour necrosis factor (TNF)-α in nonirradiated and locally ultraviolet (UV) B-irradiated nondiseased skin of patients with systemic lupus erythematosus (SLE). There were no significant differences in the cytokine mRNA expression levels in the unirradiated sites before and after 3 months of chloroquine administration. In the irradiated sites, the mRNA expression levels of all three cytokines were significantly higher than in the unirradiated group, approximately 24 h after irradiation, before chloroquine treatment. Significantly lower expressions of IL-1β, IL-6 and TNF-α mRNAs were noted in irradiated skin samples after 3 months of chloroquine treatment. These results demonstrate the local inhibitory effects of chloroquine on UVB-induced upregulation in the mRNA expression of proinflammatory cytokines in irradiated skin of patients with SLE, and provide further insight into the apparent immunomodulatory, anti-inflammatory and photoprotective properties of chloroquine. *Br J Dermatol* 2008; 159:1142–8.

Human herpesvirus 7 detection by quantitative real time polymerase chain reaction in primary cutaneous T-cell lymphomas and healthy subjects: lack of a pathogenic role

Diagnosis	HHV7 Neg,n(%)	Pos, n(%)	Total
CD30+LC	8 (57–1%)	6 (42–9%)	14
LyP	18 (75–0%)	6 (25–0%)	24
MF	71 (100%)	9 (0%)	71
SS	30 (76–9%)	9 (23–1%)	39
Total patients	127 (85–8%)	21 (14–2%)	148
HD	57 (67–9%)	27 (32–1%)	84

Viral infection has been suggested for superantigenic activation of T lymphocytes in the pathogenesis of primary cutaneous T-cell lymphoma (CTCL). Human herpesvirus 7 (HHV7), a CD4+ T-lymphotropic herpesvirus, could constitute a potential pathogenic cofactor. HHV7 DNA expression was investigated in patients with CTCL and healthy skin donors (HD) by quantitative real time polymerase chain reaction to evaluate its potential pathogenic role. Twenty-seven of 84 (32%) HD were positive for HHV7 DNA. As to CTCL, HHV7 DNA was positive in 23% of patients with Sézary syndrome, 43% with CD30+ large-cell lymphoma and 25% with lymphomatoid papulosis, while it was negative in all of 71 with mycosis fungoides. These results exclude a pathogenic role of HHV7 in CTCL, suggesting the possibility of skin as a latency site. *Br J Dermatol* 2008; 159:1149–55.